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Impact of instrumental analysis of stiff knee gait on treatment appropriateness and associated costs in stroke patients

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ABSTRACT

Background: Stiff Knee Gait (SKG) in stroke patients is typically treated by the inhibition of the rectus femoris (RF) with botulinum toxin (BoNT) after clinical evaluation, obtaining an average pooled recovery in knee flexion (KF) of 7 degrees.
Purpose: Our hypothesis is that this limited recovery after BoNT could depend on the inadequacy in the selection of patients to be treated. The aim of this study was to assess the percentage of inappropriate treatments (PIT) that can be avoided when instrumental gait analysis (GA) is used, and to estimate the associated cost savings.
Methods: We retrospectively analyzed GA data from chronic stroke patients with SKG and clinically assessed knee extensors spasticity referred to our laboratory over a five-year period. Gait kinematics and dynamic electromyography data were used. Patients were considered unsuitable for RF inhibition when: their SKG was determined by inadequate ankle push-off (APO) rather than by a brake from knee extensors, based on a previously published algorithm using gait kinematics (PIT_{KIN}); when RF was not active during KF (PIT_{EMG}); and when a proximal braking mechanism was found, if this was not due to RF activity (PIT_{GA}).
Results: 160 patients, age 20–87 years, gait speed 9–77%height/s, KF peak -4–44 degrees, were included. Of these, in 119 cases poor APO was the main cause of SKG, thus leading to PIT_{KIN} = 74%. In 48 out of 107 non-obese subjects, RF spasticity was not involved in SKG, resulting in PIT_{EMG} = 45%. Finally, patients with a braking activity as the main cause and concurrent RF activity were 20/107 = 19%, resulting in PIT_{GA} = 81%
Significance: When treating SKG, proper use of GA can reduce the percentage of inappropriate treatments by BoNT at the RF up to 81%. Savings are in the order of €100k/year when considering centers treating 100 or more patients/year.

1. Introduction

Stiff-knee gait (SKG) is a prominent feature in post-stroke walking, characterized by limited knee flexion (KF) during the swing period of gait [1,2]. This reduces gait speed, may cause toe dragging, increases the risk of falls, and compromises the stability of gait, greatly interfering with everyday activities.

SKG can either be caused by an insufficient or absent push-off at the ankle [2–4], or by an insufficient pull-off at the hip due to hip flexor weakness [5,6] and also by the presence of an abnormal braking activity of the thigh muscles, which is typically triggered by a quick knee flexion at toe-off when the push-off is maintained [3,7–9]. Since KF is a passive result of shank and thigh quick angular accelerations [5], SKG can arise independently from RF interference [10] when these mechanisms are missing.

In medical literature, overactivity of the rectus femoris (RF) muscle is often indicated as the primary cause of SKG [11,12], and historically it has typically been uniquely attributed to spastic quadriceps activity [13]. Consequently, treatments to relieve SKG focus on reducing RF interference with botulinum toxin (BoNT) injection, which is considered the gold standard procedure [14,15]. RF inhibition is chosen based on clinical assessment and in the presence of spasticity. This is assessed at the bedside by means of several scoring systems that trigger the abnormal stretch-reflex of knee extensor muscles by provoking a quick KF, such as the Modified Ashworth Scale (MAS), the Tardieu scale and the Wartenberg's pendulum test. However, during gait, spasticity may not be triggered when KF takes place slowly, as in the case of an impaired foot-ankle complex, resulting in an inadequate ankle push-off

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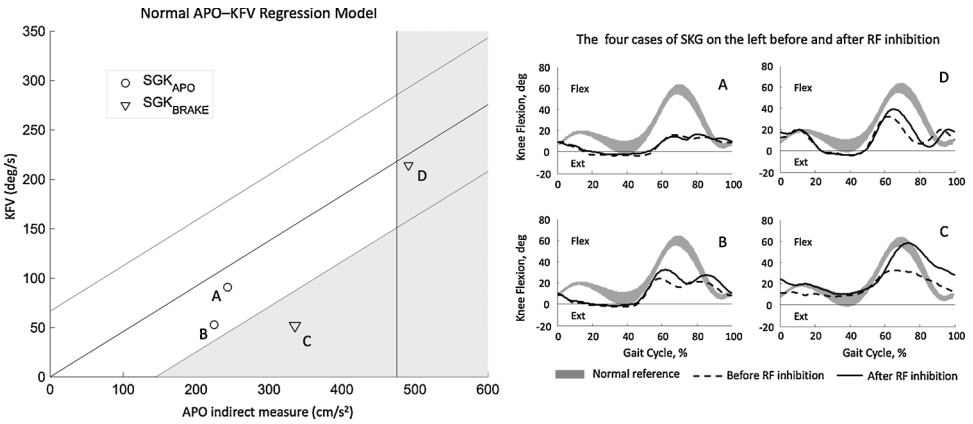

Fig. 1. The peak of vertical acceleration of the malleolus was used as an indirect kinematic-based measurement of push-off (APO). The relationship between APO and knee flexion velocity (KFV) during pre-swing was studied in healthy subjects when: walking fast; at a self-selected speed; slowly and very slowly. From a regression analysis, the APO-KFV cause-effect relationship resulted strictly linear ($R^2 = 0.967$). The regression line (continuous line) and its 95% confidence interval (dashed lines) are plotted in Fig. 1. The vertical line indicates the lower limit of normal APO. The gray area highlights the regions where patients are classified as SKG_{Brake}.
For patients with KFV congruent to APO (within the 95% confidence interval of the model) and with APO lower than its normal value (cases A and B), SKG is mainly due to the lack of APO itself (SKG_{APO}) and the improvement after RF inhibition is either null or minimal. For patients with KFV lower that the one expected from APO (below the 95% confidence interval) and with APO lower than its normal value (case C), a brake produced by the knee extensor muscles is present (SKG_{Brake}), along with a lack of APO. In this scenario, RF inhibition can be an effective tool. For SKG patients with KFV congruent to APO (within the 95% confidence interval) and APO within its normal range (case D), SKG is due to a brake from knee extensor muscles (SKG_{Brake}) that only affects swing. Also in this case RF inhibition can be effective. Noteworthy, the double bump in swing can be produced by muscles other than RF (cases B and D), such as the Vasti.

(APO) [3].

When SKG is treated by focal RF inhibition, about 80% of the cases result in a KF of less than 30 degrees, whereas the desirable target is of at least 45°, and where 60° is considered a normal value [14,16,17]. Only a few patients succeed in achieving the full range of KF during gait [14,16,17]. Our hypothesis is that this limited efficacy depends on the low appropriateness of the interventions, which are primarily focused on RF inhibition.

The instrumental assessment of gait kinematics and muscle activation can be used to determine the causes of SKG, thus facilitating the selection of the most appropriate treatment.

We recently presented an algorithm, based on instrumental gait analysis data, to differentiate among the causes of SKG - the inadequate APO and the presence of knee extensors braking activity - so as to identify a subset of patients who would benefit from focal inhibition [7]. A further increase in treatment appropriateness can be achieved by a direct measurement of the quadriceps muscles by dynamic electromyography (dEMG) [18,19].

The aim of the current study is to estimate the percentage of inappropriate SKG treatments by focal RF inhibition that can be avoided by adding an instrumental assessment of gait to the clinical evaluation. The possible impact on costs will be also addressed.

2. Methods

2.1. Study design and settings

This is an observational retrospective study that analyzed GA data from stroke patients recorded over the period of time from 2012 to 2017 at the Motion Analysis Laboratory (LAM), Neuromotor and Rehabilitation Department, Correggio (RE), AUSL-IRCCS of Reggio Emilia (Italy). Patients were referred to our service of functional assessment from neighboring hospitals and centers.

2.2. Participants

Adult stroke patients suffering from SKG were included in this study based upon the following criteria: (1) left or right hemiplegia caused by ischemic or hemorrhagic stroke; (2) chronic stroke (>12 months); (3) SKG, defined as a peak of knee flexion during swing below 45° [2]; (4) quadriceps spasticity (MAS ≥ 1); (5) ability to walk at least 10 m without shoes or orthoses; (6) walking with or without aids. The exclusion criteria were: (1) prior neurotomies to correct lower limb deformities, and (2) treatment with BoNT in the 12 months period preceding the evaluation.

2.3. Main outcome

The main outcome of this study is the percentage of inappropriate treatments (PIT) by RF focal inhibition that can be avoided by adding GA before the clinical assessment with decision making. At first, we computed PIT from kinematics data only (PIT_{KIN}), then from dEMG data only (PIT_{EMG}), and finally form a combination of the two (PIT_{GA}).

2.4. Secondary outcome

The secondary outcome of this study is the potential effect on costs consequent to the increase in treatment appropriateness. The impact of this approach on the overall cost was obtained by multiplying PIT by the standardized cost for the BoNT treatment, available from existing systematic reviews.

2.5. Identification of patients with SKG due to knee extensor braking activities by means of gait kinematics

The model described by Campanini and colleagues [7] to identify the main cause of SKG was applied to all patient data. This was obtained based on data from healthy subjects walking at spontaneous, slow and very slow speeds. It quantifies the cause-effect relationship between the vertical acceleration of the distal portion of the shank, due to APO, and the mean knee flexion velocity KFV. This relationship was strictly linear ($R^2 > 0.9$); the higher the upward acceleration, the higher the KFV. In presence of pathological mechanisms which result in a knee extension moment, the effect of APO on KFV is lower, or much lower, and patients' data fall below the normal cause-effect regression line. The model of the normal APO-KFV relationships along with its 95% confidence interval is shown in Fig. 1. The procedure used to classify patients' SKG between APO (SKG_{APO}) and proximal brake (SKG_{Brake}) is hereby explained. Data from four patients with different classifications are also presented in Fig. 1, along with one's knee kinematics before and after RF inhibition by anesthetic block of the femoral nerve branch.

In the present study, kinematic data analysis to obtain APO and KFV was performed as detailed in Table 1 using Matlab (The Mathworks Inc., Thorofare, USA).

Table 1
Data analysis procedure. The letterings d1, d2, and d3 were used in order to be consistent with prior studies relating to SKG.

Source	Protocol for data acquisition	Data analysis procedure	Outcome variables	Notes
Kinematics	- six-camera motion capture system, (SMART-DX, BTS Bioengineering, Milan, Italy); - marker placement according to the Conventional Protocol; - walking barefoot at comfortable spontaneous speed; - 5 trials were analyzed per patient.	- knee angular velocity and ankle vertical acceleration were computed from GA data by single and double differentiation, respectively, inclusive of noise smoothing; - based on foot events, data were restricted to the gait cycle of the affected limb. When multiple gait cycles were available, the central one was selected; - identification of the t1 and t2 time instants corresponding to the beginning and the end of knee flexion; - t1 was identified when knee angular velocity consistently overcame a threshold of 5 degree/sec; - t2 was identified as the first peak in knee flexion after foot-off; - d1 was the knee angle before knee flexion; - d2 was the peak of knee flexion; - d3 = d2 – d1 was the amount of degrees obtained through knee flexion.	- KFV = (d2-d1)/(t2-t1) mean knee flexion velocity, expressed in deg/s; - APO ankle push-off, assessed as the peak of vertical acceleration of the malleolus during knee flexion, expressed in cm/s^2; - KFV and APO median values over trials were computed and used in the classification procedure.	The identification of t1 as the last point where KFV > 5 deg/s before the knee flexion peak was chosen instead of simply considering the minimum during stance. This was crucial in order to obtain reliable mean KFV in patients.
EMG	- wireless EMG system (FREE-EMG, BTS Bioengineering, Milan, Italy); - disposable electrodes (ARBO H124, 2 cm interelectrode distance); - SENIAM protocol for electrode placement.	- EMG data were high-pass filtered at 10 Hz to remove baseline fluctuations; - wavelet-based time-frequency analysis was applied to data between t1 and t2; - the peak of the spectrum and its frequency f_{Peak} were computed; - when $f_{Peak} \leq 50$ Hz, data were dismissed, and the trial was removed from the analysis; - the EMG envelope was computed using a zero-lag Butterworth filter with a cut-off frequency of 25 Hz.	- RF_{EPA} Rectus femoris envelope peak amplitude between t1 and t2, expressed in μV; - RF_{EPA} median values over trials were computed and used in the classification procedure	This procedure was designed to obtain reliable results, as RF EMG data can be affected by large motion artifacts at foot-off. These are a consequence of muscle and sensor wobbling, are often not limited to low frequencies and can produce false envelope peaks.

2.6. Identification of patients with SKG due to knee extensors braking contribution based on dEMG

The presence/absence of RF activity during knee flexion when walking (RF_{on}/RF_{off}) was used as the criterion to identify patients who will/will not benefit from RF inhibition in order to relieve SKG. We defined PIT_{EMG} = $\%RF_{off}$ as the percentage of inappropriate treatments by focal RF inhibition that can be avoided by adding dEMG assessment during gait. As dEMG amplitude is affected by the thickness of subcutaneous layers when using surface electrodes [20], this analysis was restricted to non-obese subjects only, with body mass index (BMI) lower than 30 kg/m^2. According to published data, RF mechanical effect was considered negligible when the peak of the EMG envelope during knee flexion was lower than 20 μV. This amplitude was specifically selected as it is lower than the minimum value that was associated with a knee extensor moment during preswing, in the study from Nene and Colleagues [5]. To obtain the RF envelope peak amplitude (RF_{EPA}), EMG data analysis was performed as detailed in Table 1 using the EMG Easy Report software (MerloBioengineering, Parma, Italy).

2.7. Identification of patients with SKG due to knee extensors braking activity by means of both gait kinematics and dEMG

A combination of the two previous methods was used to identify the percentage of patients whose main cause of SKG was the braking activity of knee extensors including RF activity ($SKG_{Brake,RFon}$). For these patients, the focal inhibition of the RF muscle would be an appropriate relieve for SKG. Consequently, we defined PIT_{GA} = 100 - % $SKG_{Brake,RFon}$ as the percentage of inappropriate treatments by focal RF inhibition that can be avoided by means of an instrumental assessment of gait including both gait kinematics and dEMG.

2.8. Cost savings estimates

Potential cost savings were computed by multiplying the percentage of inappropriate RF treatments by the average number of patients treated by RF focal inhibition at a generic center. This was estimated based on data reported in a recent survey of 38 Italian neuro-rehabilitation units dealing with the management of post-stroke spasticity with botulinum toxin [21].

3. Results

Records from 700 patients were screened and 160 subjects were included in the study. Sample characteristics are reported in Table 2.

3.1. Contribution of gait kinematic in the decrease of treatment inappropriateness

We analyzed a total of 800 gait trials, five per patient. For each outcome variable, the median value over trials was computed and used for further analysis. Fig. 2 shows the results of the classification procedure. APO was the most frequent cause of SKG, comprising 119 subjects - about ¾ of the sample. Data from 41 patients out of 160 (26%) were classified as SKG_{Brake} because a braking contribution from the knee extensors was involved in generating SKG. These are the subjects who can get SKG relieve by treatments that remove this braking contribution, including focal inhibition treatments. Consequently, the percentage of inappropriate treatments by focal inhibition that can be avoided by adding the assessment of lower limb gait kinematics was PIT_{Kin} = 100 – 26 = 74%.

3.2. Contribution of dEMG in the decrease of treatment inappropriateness

Data from 107 non-obese subjects were taken into account. RF_{EPA} during knee flexion was lower than 20 μV in 48 (45%) subjects. For these subjects, RF spasticity was not the cause of SKG. Therefore, the percentage of inappropriate treatments by focal inhibition of the RF muscle that can be prevented by simply adding the assessment of RF dEMG during walking was PIT_{EMG} = 45%.

Moreover, the patients with absent or minimal activity (RF_{EPA} < 50 μV) were 83 (78%) in total.

Table 2
Sample characteristics of SKG patients. Values are reported as mean (SD) and range, or as count for dichotomous variables.

Characteristics	Value
Numerosity	160
Age, years	55 (14); 20 – 87
Gender female/male, no.	71/87
Affected side, left/right, no.	49/109
Weight, kg	74 (12); 44 – 109
BMI, kg/m^2	26 (4); 17 – 36
Velocity, %height/s	27 (14); 9 – 77
d1, degrees	1 (11); -23 – 28
d2, degrees	30 (12); -4 – 44
d3, degrees	28 (13); 1 – 48
KVF, degree/sec	79 (50); 12 – 274
Max KFV, degree/sec	152 (86); 15 – 458
APO, cm/s^2	213 (159); 12 – 732
RF_{EPA}, μV	36 (35); 2 – 233

Abbreviations: BMI: Body Mass Index; d1: knee angle at the beginning of knee flexion; d2: first peak in knee flexion after foot-off; d3 amount of degrees obtained during knee flexion (d3 = d2-d1); KFV: mean knee flexion velocity; max KFV: peak of knee flexion velocity; APO: ankle push-off, assessed as the peak of vertical acceleration of the malleolus; RF_{EPA}: envelope peak amplitude of the rectus femoris during knee flexion.

3.3. Contribution of a comprehensive GA in the decrease of treatment inappropriateness

Fig. 3 combines the results of the 107 non-obese subjects from both kinematic- and dEMG-based classification of SKG causes. For each patient, symbols are used to indicate the main cause of SKG between SKG_{APO} and SKG_{Brake}, and absence/presence of RF is coded as white/gray. When taking into account kinematics, 28 of 107 patients (26%) were classified as SKG_{Brake}. Of these, 8 had no activity at the RF. The percentage of patients with SKG due to a braking activity caused by RF was 20/107 = 19%. Consequently, the percentage of inappropriate treatments by focal inhibition that can be avoided by adding an instrumental assessment of SKG during walking was PIT_{GA} = 100 – 19 = 81%.

3.4. Possible impact of SKG instrumental assessment on costs

Costs associated to BoNT treatments vary substantially among countries [22]. According to literature, the average cost associated with focal spasticity treatments, comprehensive of staff costs, small equipment used and consumables, ranges between 1300 and 1600 euros/year [22], when type A botulinum toxin is used during repeated treatments [23].

A recent survey of 38 Italian neuro-rehabilitation units reported an average of 462 BoNT (type A) treatments/year/center delivered to stroke patients [21]. Of these treatments, about 230 (50%) involved the lower limb, where RF was listed as the most frequently treated muscle (percentages not reported). Therefore, an average of 100 patients/year/center treated with BoNT RF inhibition seems a reasonable estimate.

Centers treating on average cohorts of 100 patients/year for SKG relieve with BoNT can save up to €105k – €130k per year when using instrumental gait analysis to identify the main cause of SKG.

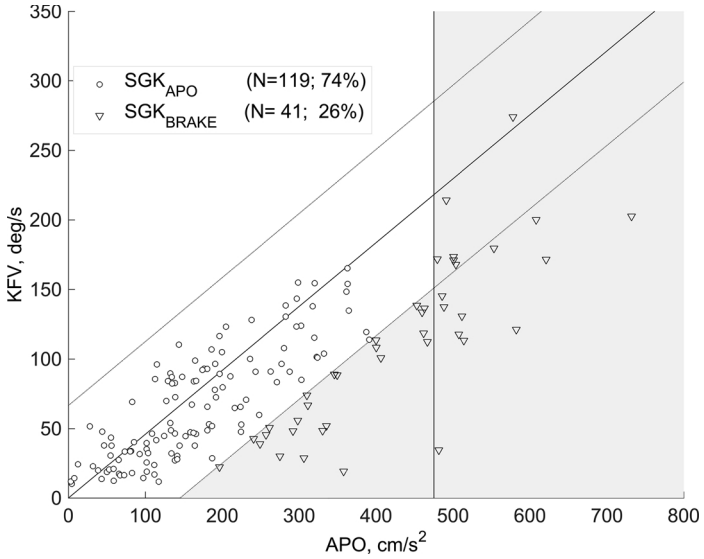

Fig. 2. Result of the classification procedure applied to data of 160 stroke patients suffering from SKG and clinically assessed spasticity of the quadriceps. Diagonal lines indicate the normal APO-KFV regression model (continuous line), along with its 95% confidence interval (dashed lines). The vertical line indicates the lower limit for normal APO. Patients with data falling in the gray area were classified as SKG_{Brake} and were represented as a triangle. Circles were used to plot data for patients classified as SKG_{APO}.

4. Discussions

For the first time in literature, this study presents the contribution of GA, inclusive of dEMG, in the reduction of the inappropriateness of SKG treatment by RF focal inhibition in order to get SKG relieve. We analyzed a sample of 160 stroke patients. The main result is that about 80% of treatments could be avoided. As illustrated in Fig. 3, only one of five patients (19%) showed RF abnormal activity as the primary cause of stiff-knee during walking, despite the presence of knee extensors spasticity during the clinical evaluation at the bedside.

However surprising it may appear, these results are entirely consistent with the percentage of patients whose SKG is resolved by RF inhibition with BotN. The review from Tenniglo and Colleagues reports a pooled increase in KF by 7 degrees [14]. The need for a better selection of patients to be treated is proven by the very large spread in the amount of KF recovery among patients, ranging from 0 to more than 20 degrees [12,16,17,24,25]. In the study from Caty and colleagues on 20 chronic stroke patients, KF increased on average by 5°, but a few patients achieved a complete relieve from SKG, with KF > 45° [16]. In the study from Stoquart on 19 chronic stroke patients with SKG, no equinus foot deformity and without knee contractures, an average 5° improvement was obtained from the treatment, including 4 non-responders [17]. In a study on 10 stroke patients, Robertson and Colleagues concluded that RF activity is only partly responsible for the decrease in knee angular velocity, as KF increased by 8 degrees after RF inhibition with BoNT, and peak knee flexion remained below normal limits for 9 subjects, with no direct relationship with the initial value [24].

Several efforts have been made to improve the efficacy of BoNT-based treatments, including ultrasound-guided injections, end-plate targeting, electrical stimulation to promote drug diffusion, use of higher dosages, and treatment sessions every three months [21,23,26]. With this study, and for the first time in literature, we suggest a further method to increase the average treatment efficacy. This is based on the *a priori* identification of subjects who would potentially benefit from the treatment.

Data in this study show that RF spasticity is not the primary cause of SKG in stroke patients. By performing a simple electromyography of the RF activity during gait the inoculation of the RF muscle can be avoided in approximately half of our sample (see Results). This result also indicates that knee extensor spasticity, as assessed by clinical examination at the bedside, cannot be used as a reliable predictor of RF spasticity during walking.

When using dEMG we always recommend to look at raw data before applying any filtering or envelope computation, as RF EMG data can be affected by large motion artifacts at foot-off consequent to muscle and

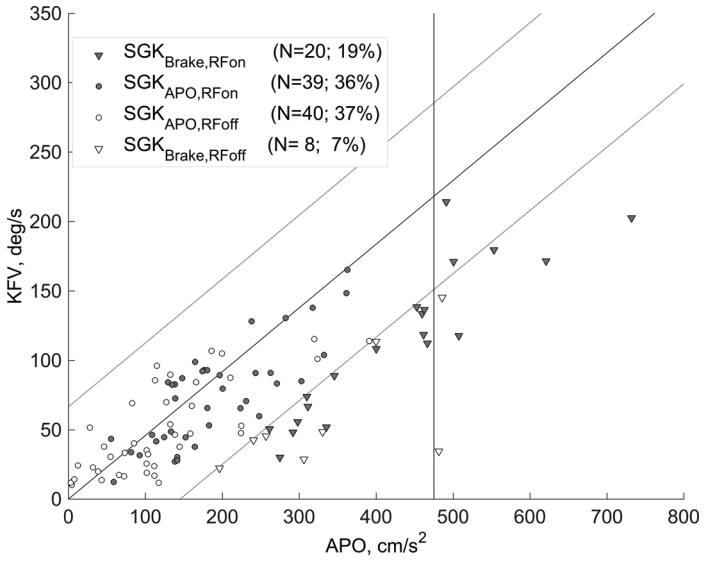

Fig. 3. Result of the classification procedure inclusive of dEMG applied to data of 107 non-obese (BMI < 30) stroke patients with SKG and clinically assessed for spasticity of the quadriceps. As in Fig. 2, circles were used for patients classified as SKG_{APO} and triangles for patients classified as SKG_{Brake}. Symbols were filled in gray when the rectus femoris envelope peak amplitude was greater than 20 μV (RF_{on}) and left white otherwise (RF_{off}).

sensor wobbling. When high-pass filtered (e.g. at 30 Hz), these artifacts may look very similar to EMG bursts and lead to the false detection of spasticity [20].

KF requires for the tibia's forward rotation over the stance foot, referred to as second rocker, followed by propulsion whilst both shank and thigh are kept within the sagittal plane of progression. When RF is active from the beginning of KF, it limits the mean KFV. In the model, subject's data lie below the confidence interval of the APO-KFV linear relationship (see Figs. 2 and 3). In this case, RF activation is triggered by muscle lengthening, irrespective of stretching speed. Conversely, when APO is not impaired, and RF is trigged later, by a velocity-dependent stretch reflex, this results in a normal APO-KFV relationship in the model, and with patient's data close to the regression line. In this case, the threshold velocity for RF reflex activation is reached after several degrees of knee flexion (i.e. at the end of stance) and the mechanical effect of RF spasticity takes place after foot-off, due to the electromechanical delay [27].

It can be noted from Fig. 3 that RF spasticity may be not the responsible of SKG even in the presence of a huge braking activity. This was the case in a subject with APO of about 500 cm/s^2 and KFV lower than 50 deg/s (Fig. 3), where RF was silent. In this case, other muscles in the quadriceps are responsible for SKG. Therefore, when a proximal braking mechanism exists, the contribution of all muscles of the quadriceps femoris, including the vastus intermedius, and the hamstrings must be measured [7,18,28]. Moreover, knee extensors shortening and increased stiffness can further limit knee flexion during swing.

4.1. Impact of GA assessment on aptness and costs

To better evaluate the possible impact of the proposed instrumental assessment on the costs of SKG management by BoNT two opposite scenarios can be considered based on clinical assessment of SKG. In the first scenario, all patients with SKG and spasticity at the quadriceps are supposed to be treated by RF focal inhibition, being RF spasticity considered the main cause for SKG (worst case scenario). In this case, about 80% of the costs could be spared, with an annual saving in the order of 100 k€/year per 100 patients.

In the second ideal scenario, all patients with preserved APO and SKG caused by a proximal brake are fully identified by the clinical assessment (best case scenario) [11]. Here, RF can be inactive in about half of the cases (45%), and BoNT injection into RF can be avoided, with a potential saving in the order of €50k/year per 100 patients.

Moreover, GA can be used to further complete the clinical assessment promoting the choice of the most appropriated treatment among rehabilitation, functional surgery, focal muscle inhibition, or no intervention at all [29].

4.2. External validity of the results

In this study we analyzed a sample of patients, whose level of impairment ranged from very severe to very mild (see Table 1), as assessed by the overall walking speed [30]. The values of self-selected walking speed, KF peak, and maximum knee flexion velocity were consistent with those collected in the review from Tenniglo and colleagues. Moreover, our sample size is about 10 times larger than that of the majority of published studies on SKG in stroke patients [14]. These considerations provide a strong external validity to our results.

4.3. Limits of the study

The main limitation of this study is that there is no outcome data on the effect of RF inhibition on SKG in the sample patients. Since our lab works as a facility for referring physician from all over the region, most of the patients included in the study were not treated by, neither prior to their arrival, nor after their assessment by our institution and we do not have access to their clinical history. To handle this limitation, we addressed two opposite scenarios in the discussions section. One being the worst-case scenario, where all patients with SKG and spasticity are treated by RF inhibition and the other, the best-case scenario, where only patients with a pathological knee extensor moment produced by the quadriceps are treated by RF inhibition.

This study analyzed a sample of adult stroke patients. Subsequently, the results could not be extended to patients with SKG affected by different pathologies.

Being a monocentric study, it could be affected by a selection bias, as patients with a very low level of impairment are seldom referred to our laboratory compared to more compromised ones.

Finally, we analyzed EMG data without any amplitude normalization, since this was not possible with the sample patients [20]. In order to counteract this source of bias, we excluded obese subjects from the analysis and set a very conservative (low) threshold of 20 μV to detect RF activity.

4.4. Conclusions

Our results indicate that the use of a proper instrumental assessment of gait can reduce from 45% to 80% the percentage of inappropriate treatments by BoNT at the RF, when targeting SKG relieve or resolution.

Ethics

The local Ethics Committee approved the study (registration number 2017/0123710).

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Declaration of Competing Interest

The authors declare that there is no conflict of interest. All authors have read and concur with the contents of the manuscript.

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